Journal of Arrhythmia 31 (2015) 116–117

Contents lists available at ScienceDirect

Journal of Arrhythmia

journal homepage: www.elsevier.com/locate/joa

Case Report

# Is it a typical crosstalk: Need for re-implantation?

Zeynettin Kaya, MD[a,*], Kursat Akbuga, MD[b], Alpay Aribas, MD[b], Ilknur Can, MD[b]

[a] Mevlana (Rumi) University School of Medicine, Department of Cardiology, Aksinne Mahallesi, 42040 Konya, Turkey
[b] Necmettin Erbakan University, Meram School of Medicine, Department of Cardiology, Turkey

ARTICLE INFO

*Article history:*
Received 9 July 2014
Received in revised form
23 July 2014
Accepted 29 July 2014
Available online 27 August 2014

*Keywords:*
Pacemaker
Dislocation
Lead switch
Safety mode
Crosstalk

ABSTRACT

With constantly evolving technology and an ever-increasing number of patients with pacemakers, clinicians will encounter various pacemaker malfunctions in their practice. While some of these issues can be solved even by using only the pacemaker's mode settings, others require re-intervention; neglecting a pacemaker's malfunction without full investigation threatens the patient's life. In this report, we describe a patient with a dual-chamber pacemaker with neglected or unresolved dyssynchronization that occurred 2 years after implantation.



## 1. Introduction

A pacemaker is a common medical device that provides electrical impulses, through electrodes, to the heart. The first device was implanted in 1958 [1], and there have been many important developments in the field since that time. Clinicians are confronted by various pacemaker malfunctions that are associated with these developments. We report a case of inappropriate pacing spikes, caused by atrial lead dislocation and subsequent engagement of the safety mode.

## 2. Case report

A 74-year-old female patient with a dual-chamber pacemaker (DDDR Pacemaker, Medtronic, Minneapolis, MN, USA) and tined lead electrodes (Medtronic, Minneapolis, MN, USA), which had been implanted 2 years previously, was admitted to the cardiology clinic with dyspnea. Physical examination was unremarkable, while electrocardiography (ECG) revealed dyssynchronization of the atrial and ventricular beats, with pacemaker spikes at the beginning and end of the QRS waves (Fig. 1A). Device interrogation revealed that the pacemaker was set to DDDR-mode, atrial and ventricular amplitude was 5.0 V, pulse width was 0.5 ms, and the atrial sensitivity had been set at 0.25 mV at presentation. The atrial pacing threshold and ventricular pacing threshold were 1.0 V and 0.5 V, respectively. The patient was pacemaker-dependent.

According to surface ECG and intracardiac ECG, atrial sensing was absent, although P waves were observed on the surface electrocardiogram. When atrial spikes occurred, the capture was ventricular, not atrial. After ventricular capture, there was an inappropriate spike at the end of the QRS wave (Fig. 2A). Therefore, atrial sensitivity was reduced to 0.18 mV. When the atrial lead was sensing appropriately, the ventricular spike occurred after an appropriate AV delay, and this resulted in ventricular capture (Fig. 2B). The patient was evaluated by using fluoroscopy, which revealed that the ventricular lead was at the correct location, although the atrial lead was in close proximity to the tricuspid valve (Fig. 3). Therefore, it appeared that the atrial lead induced ventricular capture owing to its location, and this ventricular capture was sensed by the ventricular lead, causing the pacemaker to enter the safety pacing mode. This resulted in the second spike, which came in the refractory period, and did not capture the ventricle. Eventually, the atrial lead began sensing P waves, while pacing the ventricle. This diagnosis was confirmed by using the pacemaker's AOO mode, whereby atrial stimulation resulted in ventricular capture (Fig. 2C). Ventricular capture resulted in ventricular pacing by using a pacing threshold of 0.5 V at 0.5 ms pulse width. The lack of atrial detection was resolved by programming the highest device atrial sensing parameter (0.18 mV), and the mode of the pacemaker was changed from DDD to VDD (Fig. 1B).

## 3. Discussion

In the present case, dyssynchronization between the atrial and ventricular beats resulted in abnormal pacing spikes detected on

* Corresponding author. Tel.: +90 505 2537049; fax: +90 332 322 94 19.
*E-mail address:* zeynettinkaya@yahoo.com (Z. Kaya).

http://dx.doi.org/10.1016/j.joa.2014.07.004
1880-4276/

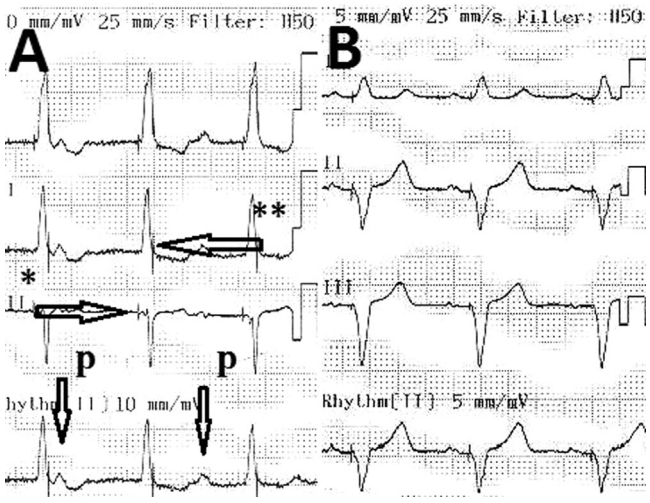


**Fig. 1.** Electrocardiography (ECG) shows dyssynchronization of atrial and ventricular beats, along with pacemaker spikes at the beginning (*) and end (**) of the QRS waves (A). ECG reveals the synchronization of ventricular-atrial beats in the VDD mode in the first half of the electrocardiogram. No under-detection was observed in the second half of the electrocardiogram, which exhibited correctly functioning VDD mode (B).

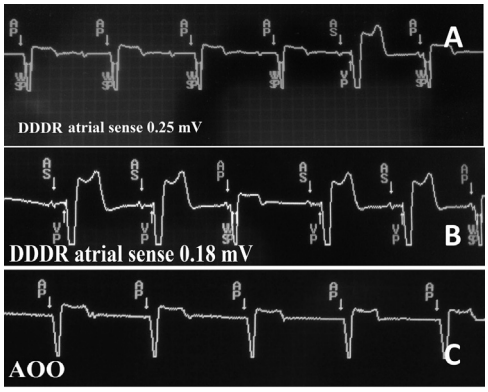


**Fig. 2.** Intracardiac electrocardiography demonstrates atrial pacing, ventricular capture, ventricular sensing, and ventricular pacing markers in order (DDDR mode, atrial sensitivity is 0.25 mV) (A). Intracardiac electrocardiography reveals that the atrial lead is sensing appropriately, the ventricular spike occurs after an appropriate AV delay, and that this results in a ventricular capture (DDDR mode, atrial sensitivity is 0.18 mV) (B). Intracardiac electrocardiography reveals steady ventricular capture in the AOO mode (C).

the surface electrocardiogram, and it is a potentially life-threatening condition. Previous case reports have suggested that this condition might be caused by crosstalk, dislocation of the leads, or inappropriate switching of the connection of the atrial and ventricular leads to the head of the generator [2–5]. Fluoroscopic examination revealed that dislocation of the atrial lead had been causing ventricular capture due to its proximity to the tricuspid valve, causing the ventricular capture to be sensed by the ventricular lead. The diagnosis was confirmed by steady ventricular capture in the AOO mode. As this was not typical crosstalk, the malfunction was resolved by programming the atrial sensing parameter and switching to the VDD mode. AV delay was kept short to prevent inappropriate ventricular spiking as well as to avoid a possible danger of R on T phenomenon caused by

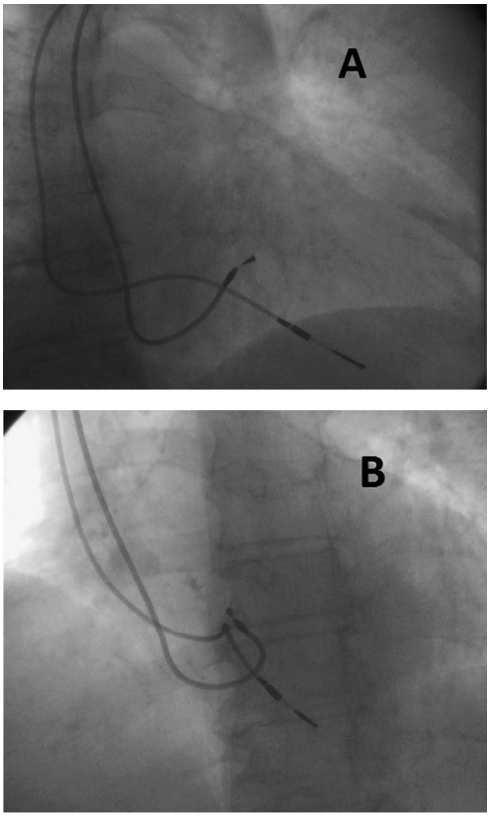


**Fig. 3.** Fluoroscopic examination reveals that the tip of the atrial lead is in close proximity to the tricuspid valve in the right anterior oblique (A) and left anterior oblique (B) positions.

sensing of the ventricular activation by the atrial lead before the ventricular lead.

In conclusion, accurate diagnosis of the malfunction allowed us to attain correct atrioventricular synchronization, without subjecting the patient to additional risks associated with invasive diagnostic and corrective procedures.

## Conflict of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and composition of the manuscript.

## References

[1] Furman S, Schwedel JB. An intracardiac pacemaker for Stokes–Adams seizures. N Engl J Med 1959;261:943–8.
[2] Lloyd MS, El Chami MF, Langberg JJ. Pacing features that mimic malfunction: a review of current programmable and automated device functions that cause confusion in the clinical setting. J Cardiovasc Electrophysiol 2009;20:453–60.
[3] Davoodi G, Faramarzi N, Shafiee A, et al. Pacemaker interrogation showing virtually no ventricular pacing in a ventricular pacing dependent patient: what is the explanation? Anadolu Kardiyol Derg 2013;13:594–5.
[4] Al Hamdi A, Jastrzębski M, Hawas JM. Atrial and ventricular lead switch at the pacemaker header: why did asystole first occur 3 years later? Pacing Clin Electrophysiol 2013;36:1431–3.
[5] Jastrzębski M. Pacemaker malfunction due to atrial and ventricular leads switched in the header: two faces of the same mistake? Pacing Clin Electrophysiol 2008;31:733–5.